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FEATURES OF ANESTHESIA IN PATIENTS WITH SPECIAL NEEDS. PART 2.

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There's nothing more poisonous than a feigned friend.
Hryhoriy Skovoroda, 18th century

Abstract. Cannabis potentially interacts with drugs commonly used for anesthesia, which can be life-threatening. Dysfunctions of various organs and systems in cannabis users classify them as patients at increased perioperative risk. When planning anesthesia, acute cannabis intoxication and chronic use should be considered, as patients with acute intoxication may require lower doses of anesthetics (but emergence from anesthesia will be significantly prolonged compared to non-intoxicated states). Chronic exposure to cannabinoids outside of acute intoxication leads to downregulation of receptors, which may result in increased anesthesia tolerance when patients abstain from cannabis before anesthesia. Regional anesthesia methods are preferred in cannabis-dependent individuals. Tolerance to propofol is increased in cannabis users. Acute cannabis intoxication mostly causes additive effects with general anesthesia drugs. Chronic cannabis use mostly results in cross-tolerance to general anesthesia drugs. Anesthesiologists, surgeons, and intensive care physicians should understand the impact of cannabis on the action of general anesthesia drugs to implement safe perioperative management.

Keywords: cannabinoids, substance interactions, local anesthesia, general anesthesia, complications, prevention.

INTRODUCTION

Cannabis is the most commonly used illicit drug with nearly 200 million consumers worldwide [1, 2].

In 2020, the UN Commission on Narcotic Drugs supported the WHO recommendation to remove cannabis from the list of particularly dangerous drugs, yet it is recognized as harmful to health and prohibited for non-medical use [3]. With the increasing prevalence of both medical and recreational cannabis use among the general population, anesthesiologists, surgeons, and intensive care physicians must understand the impact of cannabis on the human body to implement safe perioperative management [4,5].

EVIDENCE ACQUISITION

Selection articles were included for review if they (1) were published in English, Ukrainian, French, German, or Hebrew, (2) reported cannabis-related disorders affecting various organs and systems, (3) reported interactions between cannabis and drugs for local and general anesthesia, (4) informed about the clinical features of cannabis withdrawal syndrome and its treatment, (5) utilized observational study designs (cohort or crossover). A retrospective information search was conducted from 2002 to 2024 using a descriptor system-based spatial-vector model, supplemented by manual search of included articles.

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77.8 % of the literature sources used were from the last 5 years of publication, 94.4 % were from the last 10 years, and 5.6% were from earlier years of publication.

EVIDENCE SYNTHESIS

Acute preoperative cannabis use is associated with a moderate increase in the risk of perioperative complications and mortality following major elective surgeries [6]. Cannabis potentially interacts with drugs commonly used for anesthesia, which can be life-threatening [7]. Circumstances justifying postponement of surgery are crucial for anesthesiologists [4, 8]. As a preventive measure, waiting until acute cannabis effects (in intermittent users) diminish before the start of anesthesia and surgery has been proposed [9]. Cannabis use before surgery can lead to serious safety issues for both patients and medical staff. Before surgery, it is important to assess signs and symptoms of acute cannabis poisoning as it poses the greatest risk for anesthesia application. Patients with symptoms of acute cannabis poisoning are more likely to emerge from anesthesia forcibly. In individuals with a history of angina, it is important to assess cardiac function outside of angina attacks during cannabis use. Preoperative cardiac function tests and cardiology consultation may be necessary. Patients at increased risk of ischemic heart disease have an increased risk of myocardial infarction within the first hour after cannabis use, so surgeries should be postponed for at least one hour if possible [10]. Currently, there are no standardized recommendations for discontinuing cannabis use before elective procedures [11]. Despite the impact of cannabinoids on the body's organs and systems, the question remains open as to whether cannabis intake should be discontinued or continued before surgery. Current consensus-based recommendations suggest reducing cannabinoid use for 7 days before surgery to less than 1.5 g/day of smoked cannabis, 300 mg/day of cannabidiol (CBD) oil, 20 mg/day of Δ 9-tetrahydrocannabinol (THC) oil, while cautioning against use on the day before surgery and recommending limiting consumption for 6 days after elective surgery [9]. The consensus is to wait at least 72 hours from the last cannabis use before conscious sedation treatment to reduce the likelihood of drug interactions before starting anesthesia [11-14]. Recently, even more conservative recommendations have been provided, recommending cessation of oral cannabis consumption for up to 10 days [4, 15] or refraining from smoking marijuana as long as possible before surgical intervention [16].

Since cannabis is eliminated very slowly, with a tissue half-life of approximately 7 days, it can remain present in the body for many weeks after cessation of use and continue to cumulatively react to sedative agents [11, 17]. Cannabinoids are well soluble in fats, leading to slow release into the bloodstream, and a single dose is not completely cleared within 30 days [14]. This means that the cumulative dose of cannabinoids due to cessation of use may decrease somewhat, but the problem will persist, even without considering the potential development of cannabis withdrawal syndrome. Recent recommendations from the interdisciplinary network "Perioperative Pain and Dependence" suggest that patients undergoing surgery within less than 1 day have no restrictions on cannabis use due to the risk of withdrawal syndrome and increased anxiety or pain [11].

Cannabis-dependent individuals are at increased risk of developing deep vein thrombosis, pulmonary embolism, stroke, myocardial infarction, and perioperative mortality [18,19]. However, marijuana may also have antithrombotic effects [14, 20-22]. Concurrent use of cannabis with indirect anticoagulants (warfarin) and platelet aggregation inhibitors (clopidogrel) leads to supra-therapeutic increases in international normalized ratio (INR) and severe bleeding [1, 4, 10, 13, 23], so the administration of these drugs should be discontinued before surgery, and prophylaxis of thromboembolic complications should be carried out with direct anticoagulants under careful monitoring of coagulation tests and INR [10].

Severe tachycardia due to marijuana use before surgery should be controlled with beta-blockers (propranolol, labetalol, or esmolol). Marijuana may enhance the sedative-hypnotic effect of central nervous system (CNS) depressants [24,25]. Cannabis induces feelings of calmness, relaxation, and euphoria, which may seem desirable for use in anxious patients. However, in those who react naively, the opposite reaction of uncontrolled anxiety, which can escalate into a panic attack, may occur [9]. When cannabis is combined with sedative drugs such as alcohol, hypnotics, or benzodiazepines, an increased level of fatigue is observed. Concurrent use with stimulant substances such as amphetamines or cocaine may enhance the stimulating effect of these drugs [26]. Talkativeness, anxiety, and transient psychotic episodes can be treated with benzodiazepines or antipsychotic drugs [27].

Reduced immunity and susceptibility to pneumonia [28] in this population make wound infection prevention mandatory.

During cannabinoid intoxication during endoscopic procedures, there is a marked paradoxical salivary secretion, especially when propofol is used as a general anesthetic [29, 30]. However, it is desirable to avoid administering atropine for premedication, as it can cause severe tachycardia [25, 27] and paradoxical excitement [13, 31] in cannabis users. Sympathomimetics should be avoided in patients with acute marijuana use in their medical history, but

dangerous bradycardia and hypotension, which may result from high doses of marijuana, cast doubt on the necessary amount of atropine and vasopressors [14]. Therefore, anesthesiologists should exercise special caution when using intraoperative sympathomimetics and beta-blockers in those who use cannabis due to potential cytochrome P450 inhibition. Additionally, patients should be closely monitored for signs of hemodynamic instability, myocardial infarction, or stroke during surgery [10].

In the case of drug use less than 24 hours before surgery, it is advisable to consider such a patient as having a full stomach (delayed gastric emptying) [9, 27, 32-35]. Chronic marijuana users may also suffer from cannabinoid hyperemesis syndrome [36-39]. Antiemetic therapy in these individuals is generally ineffective, although some success has been reported with butyrophenones (haloperidol or droperidol) [40].

Cannabis consumption is associated with poor oral hygiene, xerostomia, caries, and periodontosis, which can have negative consequences for the airways [41]. Before intubation, a risk assessment should be conducted, focusing on potential upper airway tissue swelling and potentially increased resistance in the lower airways. Preparedness for difficult tracheal intubation due to hyperreactivity, irritation, hypersalivation, acute edema, and airway obstruction [1, 9, 10, 25, 36, 41, 42], as well as for forced changes in ventilation modes, is necessary. Preoxygenation is mandatory. The administration of dexamethasone [14, 25, 26, 43] and the addition of methylprednisolone to salbutamol in patients with partially reversible airway obstruction to reduce reflex bronchoconstriction resulting from tracheal intubation should be considered [14]. Anesthesiologists should be prepared to manage airway hyperreactivity during surgery if patients do not have secure airways due to potential irritation caused by preoperative cannabis use [10].

Hypothermia and shivering are quite commonly observed in cannabis users after awakening from anesthesia [13]. Marijuana users typically have lower fasting blood glucose and insulin levels than non-drug-dependent individuals [44, 45]. Increased heart rate, hypoxemia, oxygen delivery and consumption, myocardial ischemia, and acidosis are well-known physiological effects of shivering. The combination of these factors may lead to the development of hypoglycemic coma. Preventive and therapeutic measures may include the administration of diazepam and rimonabant [13].

Cannabis users experience reduced tear production, conjunctival injection, and decreased intraocular pressure, which collectively contribute to corneal drying [27, 36, 39, 46]. During general anesthesia in this patient population, it is important to cover the eyes with cotton pads moistened with 0.9 % sodium chloride solution.

A common mechanism of action for general anesthetics and cannabinoids is the modulation of gamma-aminobutyric acid (GABA), thus pharmacological interactions can be expected [47]. Intraoperative management of individuals who use cannabinoids is complicated by their influence on general anesthetics and the monitoring of anesthesia depth, with a simultaneous increase in potential intraoperative hemodynamic instability [36]. The most clinically significant interactions are additive pharmacodynamic interactions with simultaneous use of cannabis and other agents with similar effects [4]. An expert consensus committee agreed that when planning anesthesia, acute cannabis intoxication and chronic use should be taken into account, as patients with acute intoxication may require lower doses of anesthetic drugs (but emergence from anesthesia will be significantly prolonged compared to non-acute intoxication) [9, 40, 48].

For the anesthetic management of urgent surgical interventions in patients with acute cannabinoid intoxication, the use of a multimodal perioperative analgesic approach involving acetaminophen and a nonsteroidal anti-inflammatory drug or a specific cyclooxygenase-2 (COX-2) inhibitor in combination with local or regional analgesic techniques, if possible, is recommended [49]. Cannabinoids potentiate the action of celecoxib [32] and affect the metabolism of acetaminophen [40], and several cases of liver damage have been described with their prolonged concurrent use [36].

In patients with regular cannabis consumption, preference is given to regional anesthesia methods [16]. Dental treatment in cannabis users was accompanied by anxiety, dysphoria, and paranoia associated with distorted perception, altered relationships, and increased sensory sensitivity [9]. Adding adrenaline to the local anesthetic solution in such patients is contraindicated, as it can cause pronounced tachycardia [9, 14, 25]. The availability and clearance of bupivacaine and ropivacaine are influenced by the activity of cytochrome P450 enzymes, which depend on cannabinoid receptor stimulation [1], and the effect of cannabis on cytochrome P450 3A4 (CYP3A4) metabolism of lidocaine requires dose adjustment [48].

Several studies have reported enhanced effects of inhalational agents in animal models following the administration of synthetic cannabinoids [13]. Acute cannabis use increases the anesthetic effect and prolongs the action of ethyl ether. Experimental studies have demonstrated a decrease in the minimum alveolar concentration (MAC) of halothane in dogs by 58 %, sevoflurane by 26 %, and cyclopropane in rats

with prior administration of 2 mg/kg Δ9-THC. Another study showed a decrease in MAC for sevoflurane with intraperitoneal administration of morphine to laboratory animals at a dose of 5 mg/kg concurrently with cannabinoids. Administration of Δ9-THC orally at a dose of 10 mg/kg significantly reduces the MAC of sevoflurane in rats that have not previously received cannabinoids [36, 48]. Similar increased tolerance to inhalational anesthetics such as isoflurane and sevoflurane has been associated with cannabis consumption [13, 16]. MAC values are typically used to determine the depth of anesthesia with sevoflurane vapor, defined as the minimum alveolar concentration of volatile anesthetic that inhibits movement in response to a painful stimulus in 50 % of individuals [48]. Preoperative cannabis use by patients leads to increased tolerance to sevoflurane [10, 51, 52]. The mean total volume of sevoflurane administered during standard procedures was significantly higher in the cannabis-using group (37.4 mL vs. 25 mL, $p = 0.023$). It is known that certain anesthetic agents (opioids, propofol, benzodiazepines, lidocaine, and ketamine) can also affect the MAC of sevoflurane, so clinicians need to adjust its dose accordingly for general anesthesia [48]. In a prospective randomized study, it was demonstrated that patients receiving sevoflurane anesthesia had significantly lower levels of the endocannabinoid neurotransmitter anandamide in their blood, while no changes were detected in patients receiving propofol anesthesia [13]. Since high levels of cannabis reduce the MAC values for inhalational anesthetics in clinical settings, this phenomenon may logically prompt practitioners to establish lower levels of anesthetic vapor during the treatment of those who may be in a state of cannabinoid intoxication. This could pose significant challenges considering recent reports on the consequences of chronic cannabis use [52]. Acute cannabinoid use increases the impact of drugs that suppress cardiac activity. Increased concentrations of potent inhalational agents can lead to pronounced myocardial depression during general anesthesia [24, 25, 27, 43].

There is an antagonistic effect between Δ9-THC and propofol due to the blockade of cannabinoid receptors. Since cytochrome P-450 2B6 (CYP450-2B6) is the main pathway for hydroxylation/oxidation of propofol, increased mobilization of CYP450-2B6 after prolonged cannabis use may lead to faster propofol catabolism [29-31, 53-55], thus requiring higher induction doses for endoscopic procedures, laryngeal mask insertion, and tracheal intubation [1, 9, 10, 14, 16, 25, 26, 30, 36, 47, 56, 57]. If the bispectral index (BIS) value is above 60, induction is considered unsuccessful for this endpoint. If BIS is less than 60, a laryngeal mask attempt is made 60 seconds after propofol administration cessation, considered successful if achieved without patient response [26, 36 ,47]. A study by the American Osteopathic Association showed that marijuana users undergoing endoscopic procedures required a 200 % higher dose of propofol [30], randomized clinical trials indicate an increase in propofol dose to 5.0 vs. 3.2 mg/kg ($p < 0.025$) or to 314.0 ± 109.3 vs. 263.2 ± 69.5 mg ($p \leq 0.04$) compared to the control group in surgical interventions [1, 9, 11, 13, 47, 56, 58]. Animals simultaneously receiving cannabis and propofol demonstrated dose-dependent antagonistic interaction between tetrahydrocannabinol and propofol with significantly prolonged recovery time after anesthesia (more than 4 times longer than with propofol alone) [16, 29, 30, 53, 54]. High doses of propofol are associated with cardiovascular and respiratory system compromise [56], with arterial hypotension, bradycardia, respiratory depression, and vomiting [59]. Increased sedative doses of propofol are associated with increased brain enzyme anandamide content (a competitive inhibitor of fatty acid amide hydrolase), which degrades endocannabinoids. Propofol-induced increase in anandamide was associated with severe hypotension under anesthesia due to sympathetic response suppression mediated by cannabinoid receptor type 1 (CB1-R) and vanilloid receptor type 1 displacement. This interaction may explain one of the mechanisms of propofol-induced hypotension [13]. High doses of propofol in the presence of cannabinoids stimulate saliva production, termed «cannabis-induced hyper-salivation after propofol» (CHAP), dependent on individual metabolic genotype/phenotype, frequency of use, type of cannabis product, and time since last pill ingestion [1, 9, 16, 29, 30, 54].

Experimental and clinical studies report an additive enhancing effect of Δ9-THC, CBD, and barbiturates, inhibiting metabolism and prolonging the action of pentobarbital, sodium thiopental, pentobarbitone, and thiopentone [9, 10, 13, 16, 36, 47, 58]. There are reports of a dose-dependent antagonistic effect of Δ9-THC on the sedative effect of sodium thiopental [13].

Studies evaluating the risk of cannabis interaction with opioids mainly focus on Δ9-THC and CBD. Therefore, the potential risk of most other cannabinoids is unknown [60]. Synergy between cannabinoids and opioids, mediated by the fact that the analgesic effects of morphine are mediated by μ-opioid receptors and may be enhanced by activation of κ- and Δ-opioid receptors. Opioid and cannabinoid receptors bind to similar intracellular signaling mechanisms via G proteins, leading to decreased cyclic adenosine monophosphate (cAMP) production [61], and sedative effect may be enhanced [4, 8]. Although patients experiencing pain report stronger analgesia when using cannabis with opioids, no placebo-controlled

study has directly assessed the impact of opioids in combination with cannabis in humans [62]. Only the cannabinoid receptor CB1 is expressed in the central nervous system (CNS), recent data suggest that CB2 receptors are also expressed in the CNS, but at much lower levels and in limited areas. Both receptors are involved in pain control. It should be noted that endogenous cannabinoid ligands, namely anandamide and 2-arachidonoylglycerol, are also involved in the process. By acting through these pathways, they can influence each other and reduce pain. Cannabinoid agonists have been described to stimulate the expression of opioid precursor genes and the release of endorphins. Modulation of opioid transmission modulates some effects of the partial agonist of cannabinoid receptors CB1 and CB2 Δ9-THC on nociception. Cannabinoid antagonists suppress the ability of morphine to reduce pain [63, 64]. CBD to a greater extent than Δ9-THC, can enhance the analgesic effect of codeine, morphine, methadone, hydrocodone, and tramadol [4, 65].

Experiments on animals demonstrate an acute additive prolongation effect of acute Δ9-THC and ketamine interaction [36, 48]. Ketamine induces endogenous release of cannabinoids, which may partially explain its antinociceptive role. The psychomotor side effects of ketamine are enhanced when CBD is administered [1, 13, 66]. Tachycardia should be avoided in patients with a history of acute marijuana use, as ketamine increases heart rate [14, 25, 27, 50].

Acute cannabis intake leads to drowsiness, dizziness, and temporary impairment of sensory and perceptual functions. Marijuana can additively enhance and prolong the sedative and depressive effects of benzodiazepines [4, 8, 9, 26, 42, 47].

Although not clinically investigated, potential synergistic analgesic effects between synthetic tetrahydrocannabinol and dexmedetomidine, demonstrated in an animal model, are possible [1]. Animal studies have shown that the synthetic derivative of Δ9-THC (CP55, 940) has an additive or synergistic analgesic effect when administered concurrently with dexmedetomidine, depending on the nociceptive stimulus used [66].

Clinically observed additive effects of cannabis with phenothiazines [9, 47] and haloperidol [32].

Animal model studies and clinical observations have demonstrated prolongation of the action of propanidid and alfaxolone/alfadolone (Althesin®) with prior use of Δ9-THC [13, 36].

Activation of CB receptors leads to inhibition of voltage-gated Ca2+ channels, resulting in a synergistic effect between gabapentin and Δ9-THC [1].

Cannabinoid receptors CB1-R are in the neuromuscular junction, and there is evidence that preoperative marijuana smoking leads to enhanced acetylcholine release and increased post-synaptic action potential, although the clinical significance of neuromuscular blockers and the question of shortening the duration of muscle blockade in cannabis users are insufficiently studied [1, 10]. It is unclear whether cannabinoids are associated with the development of malignant hyperthermia, but their internal α2 activity does not rule out this possibility [46]. Pancuronium, which affects heart rate, should be avoided in patients with a history of acute marijuana use [9, 14, 27, 50].

In a double-blind randomized controlled trial, cannabis consumption before surgery was associated with increased mean intraoperative bispectral index (BIS) during anesthesia. Taking all the facts into account, cannabis consumption increased electroencephalogram (EEG) activity, making BIS a less reliable method for measuring the depth of anesthesia [36]. When monitoring anesthetic assistance, mean BIS values were higher during inpatient anesthesia in patients receiving high doses of cannabis. However, higher BIS values could be a consequence of EEG changes caused by cannabis rather than reflecting the depth of anesthesia. Therefore, the use of BIS may be inaccurate in patients with acute high-dose cannabis use [1, 13, 67].

The chronic effects of cannabinoids, beyond acute intoxication, lead to downregulation of receptors, potentially resulting in increased tolerance to anesthesia when patients abstain from cannabis prior to anesthesia [9, 40, 48]. It's worth noting that cannabinoid users are often unaware of the drug's impact on anesthesia and may withhold their specific needs, leaving anesthesiologists "working in the dark" [36]. Several clinical cases are known where patients requiring high doses of anesthesia drugs later admitted to cannabis use [47].

Despite the widespread belief in the dental community that patients with a history of cannabis use are harder to anesthetize, there is limited literature documenting this phenomenon. No significant differences were found in the effectiveness of local anesthesia, onset, or duration of action between chronic marijuana users and non-users. Overall, 88 % of non-marijuana users and 61 % of users were successfully anesthetized; however, this difference was not statistically significant ($p = 0.073$) [42]. The incidence of spinal anesthesia failure was higher among cannabis abusers compared to non-users. Dependent patients showed a slower onset and shorter duration of both sensory and motor block, requiring increased doses of anesthetics and analgesics compared to non-dependent patients [68]. No study has investigated the impact of cannabinoids on the efficacy of epidural and regional anesthesia [36].

Experimental studies in animal models and clinical settings have demonstrated cross-tolerance to barbiturates, opioids, certain NSAIDs, benzodiazepines, and phenothiazines [9, 24, 40, 47-50].

A multi-vector study revealed an increased need for higher doses of propofol and morphine in cannabis-dependent individuals beyond acute intoxication. A statistically significant difference in three types of sedation (fentanyl, midazolam, and propofol) used in cannabis users compared to non-drug-dependent individuals was identified [30, 48, 49]. Cannabis-dependent individuals exhibit a more variable response to propofol induction: they may require higher doses of propofol to achieve both loss of consciousness and adequate jaw relaxation and suppression of airway reflexes for laryngeal mask insertion [48]. Those who use marijuana daily require a higher dose of propofol than those who use it weekly or monthly [59]. The only study (47 subjects, 2 groups) did not find statistically significant differences in doses of propofol, fentanyl, and ketamine during esophagogastroduodenoscopy in the cannabis group beyond acute intoxication compared to the control group [69], but endoscopic procedures typically involve much lower doses of anesthetics compared to general anesthesia for major surgeries, and the degree of risk in this clinical scenario with a small sample size is unknown [11].

An international expert group (2021) considers cannabis use by patients in the perioperative period not contraindicated for ketamine use [40]. However, the potential psychiatric and vegetative side effects of cannabis may interfere with postoperative recovery [24].

A study conducted by the American Osteopathic Association showed that marijuana users require 20 % more midazolam and 14 % more fentanyl for conscious sedation during endoscopic procedures compared to non-smokers [1, 9, 30]. According to another clinical observation, the marijuana group required increased doses of fentanyl (0.6 vs. 0.4 mcg/kg, $p < 0.025$) for similar surgical interventions [11, 58].

Δ9-THC depletes acetylcholine stores and induces an anticholinergic effect, leading to potentiation and prolongation of the action of non-depolarizing muscle relaxants. Atracurium usually does not pose any problems related to reversal of neuromuscular blockade in such patients [13, 43].

Patients with a positive urine screening for Δ9-THC before surgery had significantly poorer pre-anesthesia health indicators and were much more likely to test positive for opioids before surgery compared to patients who tested negative for Δ9-THC [70, 71].

Cannabis use may increase the risk of perioperative airway reactivity, with an increased incidence of laryngospasm [70, 71] and bronchospasm [14, 72]. Acute cardiovascular effects include tachycardia and vasodilation, which may increase the frequency of coronary ischemia in high-risk groups [44].

The absence of standardized tools and contemporary knowledge affects anesthesiologists' ability to accurately predict the impact of cannabinoids on the course of anesthesia care for surgical interventions. The effects may be significant but nonlinear, adding another level of complexity. Anesthesia plans should be executed meticulously, considering the potential effects of cannabinoids. During treatment, the use of FDA-approved cannabinoid compounds should be continued [36].

CONCLUSIONS:

1. Disorders affecting various organs and systems in cannabis users classify them as high-risk surgical patients.
2. Regional anesthesia methods are preferred in cannabis-dependent individuals.
3. Acute cannabis intoxication mostly leads to an additive effect with general anesthesia drugs, except for propofol.
4. Chronic cannabis use generally induces cross-tolerance to general anesthesia drugs.
5. Anesthesiologists, surgeons, and intensive care physicians should understand the impact of cannabis on the action of general anesthetics to implement safe perioperative management.

Postoperative management of cannabis-dependent patients and cannabis withdrawal syndrome will be discussed in Part 3 of the literature review.

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КРАВЕЦЬ О.В., КЛИГУНЕНКО О.М., ЄХАЛОВ В.В., КОВРИГА О.В.

ОСОБЛИВОСТІ АНЕСТЕЗІЇ В ПАЦІЄНТІВ ЗІ СВОЄРІДНИМИ ПОТРЕБАМИ. ЧАСТИНА 2

Абстракт. Канабіс потенційно здатний взаємодіяти з препаратами, які зазвичай використовуються для анестезії, що може бути небезпечним для життя. Порушення з боку різних органів і систем у вживачів канабісу дозволяє віднести їх до категорії пацієнтів підвищеного операційно-наркозного ризику. При плануванні анестезії слід враховувати гостру інтоксикацію канабісом і його хронічне вживання, оскільки пацієнтам із гострим наркотичним сп'янінням можуть знадобитися менші дози препаратів для анестезії (але вихід з наркозу буде значно тривалішим, ніж поза гострою інтоксикацією). Хронічний вплив канабіноїдів поза станом гострої інтоксикації призводить до зниження регуляції рецепторів, що може спричиняти більшу толерантність до анестезії, коли пацієнти утримуються від вживання канабісу перед анестезією. Реґіонарним методам анестезії у канабіс-залежних осіб надається перевага. Толерантність до пропофолу у вживачів канабісу підвищена. Гостра інтоксикація канабісом здебільшого спричиняє адитивний ефект з препаратами для загальної анестезії. Хронічне вживання канабісу здебільшого спричиняє перехресну толерантність до препаратів для загальної анестезії. Анестезіологи, хірурги та лікарі інтенсивної терапії повинні мати розуміння впливу канабісу на дію препаратів для загального знеболювання для реалізації безпечного періопераційного менеджменту.

Ключові слова: канабіноїди, взаємодія речовин, локальна анестезія, загальна анестезія, ускладнення, профілактика.

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